Table II. Cognitive performance scores at each measurement time

	Baseline	*7 Days postoperatively*	*3 Mo postoperatively*
Fluency	25.4 ± 11.8	26.5 ± 13.2	31.2 ± 11.0
TMT Part A (s)	60.4 ± 33.7	57.6 ± 24.0	50.6 ± 18.8
TMT Part B (s)	45.0 ± 63.2	155.5 ± 79.1	134.1 ± 59.4
BVRT			
Designs correct	4.9 ± 1.9	4.1 ± 2.1	5.2 ± 1.9
Errors	8.5 ± 3.8	9.9 ± 4.9	7.6 ± 3.9
RAVLT			
Immediate memory	3.8 ± 1.3	4.1 ± 1.3	4.7 ± 1.9
Overall learning	32.9 ± 9.5	30.7 ± 8.0	35.3 ± 10.4
Retention	5.2 ± 3.5	4.8 ± 3.0	5.8 ± 3.5

Values represent mean ± SD.
TMT, Trail Making Test; *BRVT,* Benton Visual Retention Test; *RAVLT,* Rey Auditory Verbal Learning Test.

because it is not matched in terms of the characteristics of carotid artery disease.

In conclusion, tests used in this study clearly show that patients who have had a stroke differ in cognitive functioning compared with other patients undergoing CEA. These differences are noted before and after surgery. By controlling for these differences, no changes in cognitive functioning were detected that could be attributed to undergoing surgery.

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INVITED COMMENTARY

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Changes in cognitive function following carotid endarterectomy (CEA) is a difficult area in which to conduct rigorous scientific research. To date, no single study has conclusively determined whether CEA causes cognitive decline. As discussed in the preceding article, Pearson, Maddern, and Fitridge found that the severity of presenting symptom (stroke, transient ischemic attack, asymptomatic) was predictive of cognitive function scores at baseline and again three months postoperatively. Age and education level were also predictive of cognitive function. These effects were, of course, to be expected but they are nonetheless important

because they demonstrate that the chosen cognitive tests were sensitive to individual differences. For example, it is noteworthy that educational differences were still apparent despite the presenting symptom status of the patient.

When patients were retested three months after surgery, the authors observed that there were no overall differences in test scores and concluded that CEA did not cause cognitive impairment. But is this conclusion valid? If the literature on this complex subject is reviewed, the simplest conclusion to be drawn (in terms of cognitive function) is that a small number of patients improve, most stay the same, but some will become significantly worse after surgery. Patients can appear to improve for a variety of reasons. These include recovery from a premorbid cerebrovascular accident, improvement in psychological health, practice effect, greater familiarity with tasks, improved cerebral blood flow, and even inappropriate statistical analyses. Deterioration in cognitive function is classically observed in patients suffering a perioperative stroke.

However, none of the studies to date has addressed the really important question: "Can cognitive decline occur despite the patient not having suffered an operation-related stroke?" This approach requires us to explore the impact of CEA on an individual basis—ie, how often does an individual patient's cognitive function decline after CEA? Second, is the decline in cognitive function predictable? Third, if it were related to some aspect of the procedure, is there anything that might be done to prevent the secondary deterioration? If there were a hypothetical subgroup of patients likely to encounter a worsened cognitive status postoperatively, one might speculate that the most likely cause would be microembolization. This association has been evaluated and proposed in relatively small studies around the world,[1] but has never been subjected to large-scale investigation. Most importantly, if there were a causal association between perioperative embolization and cognitive decline, the adverse sequelae might be prevented by simply modifying operative technique.

Future studies on the impact of CEA and carotid angioplasty on cognitive function should clearly identify the proportion of patients suffering cognitive impairment following the operation. This could be defined in terms of a one standard deviation change or a 20% decline in at least two tests of cognitive function.[2] Cognitive improvement after CEA should be regarded as a bonus! More work is required to identify those at particular risk of cognitive decline. For some patients (and their caregivers), the latter might be just as debilitating as a stroke.

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